U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 31 May 2007

In the Matter of:

H. N.
Claimant,

CASE NO: 2006-BLA-5736

v.

J&L COAL COMPANY, INC.,
c/o OLD REPUBLIC INSURANCE
COMPANY,
Employer,

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances:

Andrew Delph, Esq.
For the Claimant

Lucy Bowman, Esq.
For the Employer

Before: LARRY W. PRICE
Administrative Law Judge

DECISION AND ORDER – DENYING BENEFITS

This is a subsequent claim for benefits under the Black Lung Benefits Act, Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. 901 *et seq.* (Act), and applicable Federal Regulation, mainly 20 C.F.R. Parts 412, 718, and 725 (Regulations).

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to coal workers' pneumoconiosis or to the survivors of persons whose death was caused by coal workers' pneumoconiosis. Coal workers' pneumoconiosis, commonly

known as black lung, is defined in the Act as "a chronic dust disease of the lung and its sequelae, including pulmonary and respiratory impairments, arising out of coal mine employment." 30 U.S.C. 902(b).

On May 19, 2006, this case was referred to the Office of Administrative Law Judges for a formal hearing. The hearing was held in Abingdon, Virginia on October 23, 2006. Neither party submitted a post-hearing brief.

At trial I admitted CX 1 through 5, EX 1 through 5, and DX 1 through 49.[1] I kept the record open in order accept post-hearing evidence from both parties. Employer objected to Miner's two interpretations of the May 30, 2006 x-ray, submitted as CX 1 and CX 2, on the grounds that Employer did not have the opportunity to have this x-ray re-read. I admitted both exhibits and allowed Employer thirty days from receipt to submit a response to this evidence. Miner was to deliver the x-ray to Employer by November 1, 2006 and Employer was to submit the re-readings of this x-ray to the Court by December 1, 2006. Miner failed to deliver the x-ray in a timely manner and so Employer requested a 30 day extension of time to submit evidence in response to this x-ray by cover letter dated November 16, 2006. The x-ray was finally exchanged by cover letter dated November 28, 2006. Employer submitted an interpretation of this x-ray by cover letter dated January 18, 2007, which I now admit as EX 6. Miner intended to submit a re-read of the November 22, 2005 x-ray, however at the time of trial, Miner had not had access to the film. I allowed Miner until December 1, 2006 to submit this evidence as CX 6; Miner failed to do so. Employer objected to the treatment records located in CX 4 and 5 on the grounds that the evidence was either duplicative or not relevant. I admitted both exhibits, but required the Miner to include specific references to the relevant treatment notes in his post-hearing brief. Miner did not file a post-hearing brief, however I will still consider all relevant treatment records located in these two exhibits.

Finally, Dr. Rasmussen's report dated May 30, 2006, submitted by Miner as CX 1, included results from a pulmonary function study and a blood gas study. Miner designated results from two other pulmonary function studies as initial evidence, and so the results of the pulmonary function study included in Dr. Rasmussen's 2006 report would exceed the evidentiary limitations set forth in 20 C.F.R. § 725.414 (2004). I therefore exclude these results. Miner only designated results from one arterial blood gas study as initial evidence, and therefore the results from the arterial blood gas study included in Dr. Rasmussen's report would not exceed the evidentiary limitations. I therefore admit these arterial blood gas study results.

PROCEDURAL HISTORY

Miner filed his first claim for benefits on March 18, 1993, which was withdrawn on February 17, 1994 pursuant to an Order issued by Administrative Law Judge Fletcher E. Campbell, Jr. (DX 1 at 144). Miner filed a second claim on February 21, 1995 (DX 2 at 380), which was granted by the District Director on November 3, 1995. (DX 2 at former 65-33).

[1] The following abbreviations have been used in this decision: DX – Director's Exhibit; EX – Employer's Exhibit; CX – Miner's Exhibit; TR – Transcript of the January 10, 2006 hearing; BCR – Board certified radiologist; and B – B-Reader.

Employer appealed and Administrative Law Judge Pamela Lakes Wood issued a Decision and Order Denying Benefits on June 30, 1997. (DX 2 at former 65-50). On May 26, 1998, the Benefits Review Board issued a Decision and Order affirming Judge Wood's decision. (DX 2 at former 65-57). Miner requested modification[2] on April 19, 1999 (DX 2 at former 65-58), which was denied by the District Director on June 17, 1999. (DX 2 at former 65-63). Miner filed a third claim for benefits on July 6, 2000[3] (DX 3 former DX 2), which was ultimately denied by Administrative Law Judge Linda S. Chapman on November 19, 2003. (DX 3 at 1005). Miner filed the instant claim on February 12, 2005. (DX 5). The District Director issued a Proposed Decision and Order, dated January 19, 2006, awarding benefits in the amount of $6190.80 initially, and $562.80 a month thereafter. (DX 37). Employer requested a formal hearing on January 27, 2006. (DX 40).

APPLICABLE STANDARDS

Miner's previous claim for benefits was denied by Judge Chapman on November 19, 2003. (DX 3 at 1005). Miner filed the current claim more than a year later on February 12, 2005 (DX 4), and therefore this claim constitutes a "subsequent claim" under the regulations. The provisions of § 725.309(d) apply to subsequent claims and are intended to provide relief from the traditional notions of res judicata. Under § 725.309(d), subsequent claims must be denied unless the evidence demonstrates that one of the applicable conditions of entitlement has changed since the prior denial. 20 C.F.R. § 725.309(d). The rulings of the United States Court of Appeals for the Fourth Circuit control in the adjudication of this case. Since this claim was filed after January 19, 2001, the regulations contained in 20 C.F.R. Part 718[4] as amended in 2001 are applicable.

To establish entitlement to benefits under this part of the regulations, a miner must prove by a preponderance of the evidence that he has pneumoconiosis, that his pneumoconiosis arose from coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis. 20 C.F.R. §725.202(d); Anderson v. Valley Camp of Utah, Inc., 12 BLR 1-111, 1-112 (1989). In Director, OWCP v. Greenwich Collieries, et al., 114 S. Ct. 2251 (1994), the U.S. Supreme Court stated that where the evidence is equally probative, the miner necessarily fails to satisfy his burden of proving the existence of pneumoconiosis by a preponderance of the evidence.

[2] Miner actually sent a letter requesting the addition of new medical evidence to the record. This letter was interpreted as a request for modification. (DX 2 at former 65-58).

[3] The claim application was originally dated March 2, 2000, which was crossed out and replaced with July 6, 2000. Miner originally filed a claim on March 2, 2000, but withdrew the claim and re-filed on July 6. Employer contended that Miner's decision to withdraw was influenced by ex parte communications between Miner and the District Director, whereby the District Director informed Miner of the date of the denial of the prior claim and advised Miner that he would receive another Department of Labor sponsored physical exam if the new claim were filed more than a year from the denial of the prior claim. Employer reported that the District Director removed references to said ex parte communications upon preparation of the file for my hearing. I acknowledge Employer's complaint and have included the removed references in the record.

[4] All of the regulations cited in this decision are contained in Title 20 of the Code of Federal Regulations.

The initial analysis is limited to a review of the condition or conditions of entitlement upon which the prior denial was based. If a miner establishes the existence of an element previously adjudicated against him, only then must the administrative law judge consider whether all the evidence of record, including evidence submitted with the prior claim, supports a finding of entitlement to benefits. 20 C.F.R. § 725.309(d)(3). In the denial of Miner's prior claim, Judge Chapman found that Miner's totally disabling respiratory impairment was not caused by pneumoconiosis.[5] (DX 3 at 1005). Therefore, if the newly-submitted evidence establishes that Miner's pneumoconiosis caused his totally disabling respiratory impairment, then I must review the entire record to determine entitlement to benefits.

ISSUES[6]

The following issues remain for resolution:

- Miner: whether Claimant was a miner as defined by the Act
- Length of employment: the number of years Claimant worked in coal mines
- Responsible Operator: whether named employer is the responsible operator under the Act
- Subsequent Claim Threshold: whether the new evidence establishes the existence of an element previously adjudicated against Miner.
- Causation: whether pneumoconiosis was a substantially contributing cause of Miner's totally disabling respiratory impairment.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

Miner was born on June 14, 1947 and currently lives in Tazewell County, Virginia. (DX 5, DX 40). Miner was married twice before; he and his first wife divorced on June 20, 1997 and he and his second wife divorced on February 29, 2000. (DX 11). On his application for benefits, Miner reported having a son that was 18 or older and disabled.[7] (DX 5). Miner reported working for numerous coal companies in various positions including, "cut machine helper", "belt pinner" and "boss". He shot coal and loaded the "machine tracker pulling coal." His last position was foreman. (DX 5).

[5] Judge Wood had found that Miner suffered from pneumoconiosis, that Miner was totally disabled and that Miner's disability was not caused by pneumoconiosis. (DX 2 former 65-50). The claim before Judge Chapman was also a subsequent claim, so Judge Chapman initially had to determine whether pneumoconiosis caused Miner's respiratory impairment. She found that Miner's impairment was not caused by his pneumoconiosis, and denied benefits. (DX 3 at 1005).

[6] The Employer objected to the District Director's practice of renumbering previously organized files, and in doing so, removing the prior references to evidence and essentially "un-numbering" the Director's Exhibits from the prior claims associated with the instant claim. I have chosen not to remand this case to the District Director due to the delay that would cause to the issuance of a decision. Instead, I have attempted to be as specific as possible in my references to evidence from prior claims.

[7] Miner submitted proof of divorce from two prior wives, and is not currently married. (DX 11). The only issue regarding dependency involves the status of Miner's son. Judge Chapman previously found that Miner's son was not a dependent under the Act. (DX 3, folder 3). A letter, dated February 15, 2005, was sent to Miner requesting proof of disability of Miner's alleged disabled child. (DX 12). Miner failed to respond and therefore has not carried his burden of proof to establish the dependency of an individual under the Act.

The record contains varied statements regarding Miner's smoking history. In the 1996 proceeding before Judge Wood, Miner testified that he had started smoking when he was 18 and quit in 1993. He stated that he had smoked an average of one pack per day yielding a smoking history of 28 pack years.[8] (DX 2). Miner informed Dr. Castle in 2003 that he smoked from age 14 or 15, stopped two or three years prior to this exam and he smoked about a half pack a day, giving him at least an 18 pack year smoking history. (DX 3). Based upon the 2006 exam, Dr. Rasmussen reported that Miner had smoked between one and one and a half packs of cigarettes per day from 1963 until 2004, which would yield a 50 pack year smoking history. (CX 1). In the report dated May 10, 2005, Dr. Rasmussen reported that Miner had smoked regularly at 16 and smoked a half a pack per day until he quit forty years later in 2003. (DX 14). Dr. Robinette commented throughout the treatment records that Miner continued to smoke despite his pulmonary impairment, however Dr. Robinette noted that miner quit smoking in a report dated 9/20/04. (CX 4). Miner testified in 1996 that he had smoked a pack per day for 28 years. The evidence indicates that Miner continued to smoke approximately half a pack per day until 2004, yielding an additional 4 pack years. I find that Miner had a 32 pack year smoking history.

Miner

Miners who establish the applicable elements of entitlement may receive benefits under the Act. 30 U.S.C. § 901(a); 20 C.F.R. § 718.1(a) (2003). A "miner" is defined as "any person who works or has worked in or around a coal mine or coal preparation facility in the extraction, preparation, or transportation of coal...." 20 C.F.R. § 718.202(a) (2003). The regulations provide a rebuttable presumption that "any person working in or around a coal mine or coal preparation facility is a miner." Id. In this case, Miner worked for various periods of time for several coal companies. (DX 9). In the proceeding before Judge Wood, Miner testified that he worked more than 23 years underground. (DX 2 former 65-48). The record contains no evidence to rebut the presumption that Miner is a miner. Accordingly, I find that Miner is a miner under the Act.

Length of Coal Mine Employment

The duration of a miner's coal mine employment is relevant to the applicability of various statutory and regulatory presumptions. Miner bears the burden of proof in establishing the length of his coal mine work. Shelesky v. Director, OWCP, 7 BLR 1-34, 1-36 (1984); Rennie v. U.S. Steel Corp., 1 BLR 1-859, 1-862 (1978). On his application for benefits, Miner alleged twenty four years of coal mine employment. The evidence in the record includes a Social Security Statement of Earnings encompassing the years 1962 through 2004, applications for benefits, Miner's summary of employment history and Miner's testimony in prior proceedings. (DX 2 former 65-48; DX 3, DX 5; DX 6; DX 9).

The Act fails to provide specific guidelines for computing the length of a miner's coal mine work. However, the Benefits Review Board consistently has held that a reasonable method of computation, supported by substantial evidence, is sufficient to sustain a finding concerning the length of coal mine employment. Croucher v. Director, OWCP, 20 BLR 1-67, 1-72 (1996)

[8] A "pack year" is the equivalent of smoking one pack of cigarettes per day, or 20 cigarettes per day, for one year. Therefore a 50 pack year smoking history could either mean the individual smoked one pack per day for 50 years, or two packs per day for 25 years.

(en banc); Dawson v. Old Ben Coal Co., 11 BLR 1-58, 1-60 (1988); Niccoli v. Director, OWCP, 6 BLR 1-910, 1-912 (1984). Thus, a finding concerning the length of coal mine employment may be based on many different factors, and one particular type of evidence need not be credited over another type of evidence. Calfee v. Director, OWCP, 8 BLR 1-7, 1-9 (1985).

Upon review of the record in this case, it is initially noted that the District Director found 22 years of coal mine employment, starting on approximately June 1, 1947 and ending December 17, 1991. (DX 37). Miner testified that he worked in the mines for 24 years. (DX 2 former 65-48). Based upon my review of the record, I place the greatest weight on the Social Security records because they are documented, independent evidence of Miner's coal mine employment. The regulations provide that a miner who has worked in or around a coal mine for 125 days during a calendar year, has worked one year of coal mine employment. 20 C.F.R. § 725.101(a)(32)(i) (2003).

The record does not contain information regarding the beginning and ending dates of Miner's coal mine employment. Section 725.101(a)(32)(iii) provides a formula to determine a miner's length of coal mine employment. The formula uses the miner's yearly income divided by the average daily wage for coal miners in that particular year. The average daily wage is taken from statistics reported by the Bureau of Labor Statistics. This formula will be utilized in this claim as the beginning and ending employment dates are not of record.

Miner worked more than 125 days per year in coal mine employment for the years 1968 through 1987 and 1991 through 1992. Therefore, pursuant to § 725.101(a)(32)(i), I credit Miner with one year of coal mine employment for each of those years, which totals 22 years. Miner worked less than 125 days per year for the years 1967, 1988 through 1989 and 1992. The regulations provide for the calculation of a fractional year of employment amounting to less than 125 days in a calendar year. 20 C.F.R. § 725.101(a)(32)(i) (2003). Miner's qualifying employment for the aforementioned fractional years totals 1.98 years. In sum, Miner has worked a total of 23.98 years in qualifying coal mine employment.[9]

Responsible Operator

In order to be deemed the responsible operator liable for the payment of benefits, an employer must have been the last employer in the coal mining industry for which the miner had his most recent period of coal mine employment of at least one year, including one day after December 31, 1969. 20 C.F.R. §§ 725.492(a), 493(a) (2004). In England v. Island Creek Coal Co., 17 B.L.R. 1-141 (1993), the Board emphasized that it is the Director's burden to investigate and assess liability against the proper operator. Then the regulatory amendments at 20 C.F.R. §725.495(c)(2) (2001) shifted the burden to require that the designated responsible operator establish "[t]hat it is not the potentially liable operator that most recently employed the miner."

[9] Section 725.101(a)(32)(iii) requires that if the formula is used to calculate length of coal mine employment a copy of the Bureau of Labor Statistics table must be incorporated into the record.. I have included this table in the record as Appendix A.

In the proceeding before Judge Wood, Miner testified that his last job in coal mine employment was with J & L Coal Co. for a period of two years. (DX 2 former 65-48 at 10). This contention is supported by the Social Security records. (DX 9). Therefore, I find that J & L Coal Co. is properly named as the responsible operator in this claim.

NEW MEDICAL EVIDENCE[10]

X-ray Reports

Exhibit	Doctor	Qualifications	Date of X-ray	Date of Reading	Film Quality	Interpretation
CX 4	McReynolds	BCR[11]	8/29/06	8/30/06		Pulmonary hyperinflation consistent with COPD with scarring in the upper lobes. Nodular interstitial lung disease in the upper lobes consistent with reported history of CWP. Lungs are hyperinflated but free of an acute process. There is parenchymal scarring. No pleural effusions.
EX 6	Wiot	B/BCR	5/30/06	12/11/05	1	Negative for CWP, em, fr
CX 1	Rasmussen	B	5/30/06	5/31/06	1	1/1, p/s, A, em, hi
CX 2	DePonte	B/BCR	5/30/06	8/14/06	1	1/1, s/r, A, ax, fr
EX 5	Castle	B	11/22/05	11/24/05	1	1/1, t/s, bu, em, tb. Changes do not look like CWP. Changes are of granulomatous disease.
CX 5 at 83	Eryilmaz	A[12]/BCR[13]	8/2/05			There is interstitial fibrosis, emphysema and some infiltration in the right upper lobe. There are bullas and possible broncheictasis in the right upper lobe. There is

[10] "A subsequent claim shall be processed and adjudicated in accordance with the provisions of subparts E and F of this part, except that the claim shall be denied unless the claimant demonstrates that one of the applicable conditions of entitlement (see Secs. 725.202(d) (miner), 725.212 (spouse), 725.218 (child), and 725.222 (parent, brother, or sister)) has *changed since the date upon which the order denying the prior claim became final*." 20 C.F.R. §725.309(d) (2001). (emphasis added). Both parties submitted evidence that was in existence prior to Judge Chapman's decision, issued on November 19, 2003. I admitted and reviewed this evidence; however I have not summarized this evidence here because it is not probative under my analysis of the subsequent claim threshold because "the subsequent claim may be approved only if *new evidence* submitted in connection with the subsequent claim establishes at least one applicable condition of entitlement." § 725.309 (d)(3). (emphasis added). Employer designated the following as initial evidence for this subsequent claim: Dr. Wiot's interpretations of the March 26, 2003 x-ray and CT scan, the pulmonary function studies and arterial blood gas studied conducted by Dr. Fino on October 30, 2000, and the medical reports completed by Dr. Castle and Dr. Fino on March 26, 2003 and October 10, 2000 respectively. Claimant designated the following as initial evidence for this claim: pulmonary function studies conducted by Drs. Rasmussen and Robinette, dated September 25, 2000 and August 1, 2002;,the blood gas study conducted by Dr. Rasmussen on September 25, 2000 and the medical report completed by Dr. Rasmussen on September 25, 2000. Also numerous treatment records were admitted by the Claimant that predate the prior claim.

						osteoporosis and kyphosis. No other abnormality is seen. The impressions include COPD and old TBC in the right upper lobe. Activity could not be determined.
EX 1	Wiot	B/BCR	5/10/2005	7/30/05	1	Negative for CWP, bu, em, fr; stranding extending from the right hilum
DX 14	Rasmussen	B	5/10/05	5/13/05	2	1/0, s/s, A, em
CX 4	Radhakrishnan	BCR[14]	9/14/04	9/16/04		There are chronic interstitial changes which are unchanged from the prior examination. There is also an ill defined opacity in the RUL which is stable from the prior study.
CX 4	Patel	A[15]/BCR[16]	8/19/04	8/23/04		Comparison with 8/16/04 study. Extensive interstitial changes as well as small subcutaneous emphysema for the right lower chest wall. From the available radiographs in the expiratory phase, no definite pneumothorax identified.
EX 4	Hurt		8/16/2004	8/16/2004		There remains right apical and lateral pleural thickening presumed significant fibrosis and scarring of the right upper lobe with mild secondary volume retraction of the mediastinal structures to the right. The lungs are hyperinflated with changes of chronic obstructive and interstitial pulmonary disease. There is minimal thickening of the right fissure unchanged. Removal of the right chest tube with minimal pneumothorax. Stable diffuse lung changes as reported. Stable subcutaneous emphysema of the right chest wall.

[11] I take judicial notice of board certification in radiology. The American Board of Medical Specialties provides this information at http://www.abms.org/searchdetail.asp?key=61893.

[12] I take judicial notice of A-reader certification. This physician was identified as a A-reader in the NIOSH Comprehensive Reader List found at: http://www.oalj.dol.gov/PUBLIC/BLACK_LUNG/REFERENCES/REFERENCE_WORKS/BREAD3EF_08_05.HTM.

[13] I take judicial notice of board certification in radiology. See note 11.

EX 4	Hurt		8/15/2004	8/16/2004	Stable chronic diffuse interstitial lung disease and emphysema with significant presumed scarring and pleural thickening of the right upper lobe with mild volume retraction of the right hilar structures. Probable stable retrocardiac atelectasis.
EX 4	Kennedy Jr.	B/BCR	8/13/2004	8/13/2004	Chest tubes in place with no evidence of residual pneumothorax. No change in the pulmonary status when compared with recent films with exception of questioned peribronchial infiltrate or atelectasis in the retrocardiac space. The area of density previously described in the right subapex and extending toward the right hilum is noted. The lateral film also suggests some peribronchial or linear atelectasis in the left lung base in the retrocardiac space.
EX 4	Kennedy Jr.	B/BCR	8/11/2004	8/11/2004	(portable) There is some linear density now seen in the left lung base consistent with linear atelectasis. No change in the cardiopulmonary status from the study of the previous day. Significant subcutaneous emphysema. Small localized area of pneumothorax within the periphery of the right apex.
EX 4	Kennedy Jr.	B/BCR	8/10/2004	8/10/2004	(portable) Area of density previously described is noted. There may be a small localized area of pneumothorax adjacent to this area of increased density. Minor fissure is somewhat more prominent than on the study of 8/5/04 suggesting the possibility of some increasing right pleural effusion. Increasing subcutaneous emphysema.
EX 4	Kennedy Jr.	B/BCR	8/6/2004	8/6/2004	(portable) The area of density in the

[14] I take judicial notice of board certification in radiology. See note 11.
[15] I take judicial notice of A-reader certification. See note 12.
[16] I take judicial notice of board certification in radiology. See note 11.

					periphery of the right subapex has decreased somewhat from recent film and perhaps represents either infiltrate or atelectasis. No new abnormalities are seen. No residual pneumothorax seen. Some improvement in the area of density in the periphery of the right subapex.
EX 4	Hassett	B[17]	8/5/2004	8/5/2004	Right-sided pneumothorax involving predominantly the mid and lower portion of the right lower lung fields. Some increased density in the periphery of the right apex and in the right lung base felt to probably represent compression atelectasis. The left lung appears to be fully expanded and free of active or acute disease.
EX 4	Kennedy Jr.	B[18]/BCR[19]	8/5/2004	8/5/2004	(portable) The pneumothorax appears to have resolved but there continues to be density seen in the right apex extending from the right hilum.

Pulmonary Function Studies[20]

Exhibit #	Physician	Date of Study	Tracings Present?	Flow-/olume oop?	Broncho-dilator?	FEV 1	FVC/ MVV	Age/ Height	Qualify?	Coop and Comp. Noted
EX 5	Castle	11/22/05	Yes	Yes	Yes	1.37/ 1.51	3.50/ 3.77/ 56	58/ 65	Yes/ Yes	Yes
DX 14	Rasmussen	5/10/05	Yes	Yes	Yes	1.88/ 2.07	4.64/ 4.75	57/ 64	No/ No	Yes

[17] I take judicial notice of B-reader certification. This physician was identified as a B-reader in the NIOSH Comprehensive Reader List found at: http://www.oalj.dol.gov/PUBLIC/BLACK_LUNG/REFERENCES/REFERENCE_WORKS/BREAD3EF_08_05.HTM.

[18] I take judicial notice of B-reader certification. See footnote 17.

[19] I take judicial notice of board certification in radiology. See note 11.

[20] 20 C.F.R. 718 Appx. B establishes the standards for the administration and interpretation of pulmonary function tests.

Arterial Blood Gas Studies[21]

Exhibit #	Physician	Date of Study	Altitude	Resting (R) Exercise (E)	PCO2	PO2	Qualify?	Age	Comments
EX 5	Castle	11/22/05	Not listed	R	36.3	57.7	Yes[22]	58	Resting ABGs show hypoxemia. Carboxyhemoglobin level is normal.
DX 14	Rasmussen	5/10/05	0 to 2999	R E	32 23	66 68	Yes/ Yes	57	
CX 1	Rasmussen	5/30/06	0 to 2999	R E	30 30	69 50	Yes/ Yes	58	

Biopsy Evidence

Exhibit Number	**Physician**	**Date of Biopsy Report**	**Comments**
EX 2	Naeye	3/27/2006	Based upon four slides. The slides contained multiple, very small pieces of tissue, some of which could be interpreted as coming from the lung. The largest piece was 8.5 x 2 mm, although most pieces are much smaller. "Much of the tissue available for microscopic review is comprised of old fibrosis with a small amount of black pigment and just a few birefringent crystals tiny enough to have been fibrogenic. Thus, enough samples of lung tissue are available for review to make the diagnosis of very mild, simple coal workers' pneumoconiosis. No lesion has anywhere near the size (2.0 cm) or the microscopic characteristics required to make the diagnosis of complicated CWP."
CX 3	Shilo	8/20/04	Diagnosis: anthracosilicotic nodules, consistent with CWP. Sections of lung reveal confluent anthracosilicotic nodules accompanied by emphysematous changes and scarring. We agree that due to severe freezing artifact observed on your

[21] 20 C.F.R. 718 Appx. C establishes the standards for the administration and interpretation of arterial blood gas studies.

[22] These results qualify for the first two altitude groups, but does not qualify if the altitude of the location of testing exceeds 6000.

			frozen sections there is a prominence of type 2 pneumocytes. No significant cytological atypia is present on the permanent sections to justify a diagnosis of bronchioloalveolar carcinoma, however. Considering the provided clinical history, these histological findings fit well with a diagnosis of coal workers' pneumoconiosis. The confluent nature of the fibrosis and reported localized fibrosis on radiological studies raise the concern of progressive massive fibrosis (PMF). The limited biopsy sample makes us reluctant to render that diagnosis, and correlation with radiological findings could be helpful in evaluating the extent of this lesion. No microorganisms are found on GMS and AFB stained sections.

Other Medical Evidence

Exhibit #	Physician	Type of Record	Date of Report	Summary
CX 4	Bergren	CT scan	9/1/05	Indication: follow up progressive massive fibrosis, recurrent hemoptysis, COPD. Compared to the 4/25/05 exam, there has been no significant interval change in fibrotic changes in the right upper lobe with calcifications. Patchy areas of fibrosis consistent with the diagnosis of progressive massive fibrosis are also present in the left upper lobe and superior segments of the left lower lobe again not significantly changed as compared to prior study. A few pleural plaques are present bilaterally. Impression: stable fibrotic changes in the right greater than left upper lobes consistent with progressive massive fibrosis. Stable diffuse emphysematous changes throughout the bilateral lungs.
CX 4	McReynolds	CT scan	4/25/05	Comparison to the 12/20/05 exam. Indication "F/U progressive massive fibrosis." There is no axillary, mediastinal, hilar, or subarinal adenopathy. There are no pleural or pericardial effusions. There are severe panlobular emphysematous changes with bullous disease in the upper lobes. There are also areas of centrilobular emphysematous changes in the lower lobes. There is chronic scarring/fibrosis in the right upper lobe with internal calcifications. There are

				scattered areas of scarring throughout both lungs. There is a stable 5mm subplearal nodule in the posteromedial aspect of the left upper lobe. Impression: Severe panlobular and centrilobular emphysematous changes. Stable scarring and/ or fibrosis in the right upper lobe with interspersed calcifications.
CX 4	Radhakrishnan	CT scan	12/20/04	Indication: pulmonary fibrosis. Comparison to the 6/12/03 study. There has been interval progression of scarring in the right apex since the prior examination. This area of scarring appears to have developed some central calcifications since the prior examination. There is advanced bullous emphysematous disease throughout the lung fields, which is unchanged from the prior exams. No significant interval change in previously noted nodes within the paratracheal and prevascular spaces. There is a small nodal area within the subcarinal region which is unchanged since the prior exam. Impression: interval progression of scarring and fibrosis in the right apex since prior study of 6/12/03. There also appears to have been interval development of calcifications within this area of scarring since the prior exam. Advanced bullous emphysematous disease with subpleural nodules which are unchanged from the prior exam.
EX 4	Hassett	CT scan	8/5/04	A right-sided chest tube is noted. There is a small residual anterior pneumothorax present. There is a focal irregular fibrotic area seen in the mid posterior aspect of the right upper lobe. The density continues to the pleural surface laterally. There are several small bullae seen associated with this density. Minimal chronic interstitial changes are present in the right base posteriorly. No hilar or mediastinal lymphadenopathy is noted.
EX 1	Wiot	CT scan	3/26/03	There is no evidence of CWP. There are bullous changes in both apices, greater on the right than on the left. The lung fields show severe emphysema. There is linear fibrotic stranding at the right apex with associated pleural disease. Pleural disease is not a manifestation of coal dust exposure. This almost assuredly is a post-inflammatory process. *He also noted that CT is medically acceptable for evaluation of pulmonary problems and is beneficial in confirming or denying the presence of CWP and can also be beneficial in recognizing complicated pneumoconiosis when it is not evidence on routine x-rays.

Medical Reports

Dr.D.L. Rasmussen (CX 1)

Dr. Rasmussen completed a report dated May 30, 2006. Dr. Rasmussen noted that Miner had smoked between one and one and a half packs of cigarettes per day from 1963 until 2004. Dr. Rasmussen recorded an occupational history of 24.5 years in the coal mining industry. Dr. Rasmussen listed the details related to Miner's numerous duties in the mines. Dr. Rasmussen diagnosed both simple and complicated pneumoconiosis.

Dr. Rasmussen reported that Miner had experienced shortness of breath on exertion for more than 15 years, had difficulty during the last years of his mining work, could walk 160 feet on level ground at a slow pace, could climb a flight of stairs if he does so slowly. Dr. Rasmussen noted that Miner had a "chronic, morning productive cough," wheezed in the morning, at night and on exertion, slept on two pillows, and coughed small amounts of blood on several occasions. He had pneumonia numerous times, and reported having "pleurisy in the remote past." Miner had been told that he had emphysema, COPD and CWP based on a lung biopsy conducted in 2004. Dr. Rasmussen also noted that Miner had a spontaneous pneumothorax in 2004 that necessitated a biopsy, which was first thought to yield evidence of cancer, however the Armed Forces Institute of Pathology "reported no malignancy, but evidence of anthricosilicotic nodules consistent with [CWP]." (CX 1). Miner complained of "right upper anterior sharp pains" and has been hospitalized in the past for chest pains.

Dr. Rasmussen performed a physical examination. He found that Miner's breath sounds were markedly reduced and Miner had a prolonged expiratory phase with forced expirations; however there were no rales, rhonchi or wheezes. (CX 1). Dr. Rasmussen also based his conclusions on the x-ray film and laboratory studies obtained in conjunction with the physical examination. He reported that the x-ray was positive for pneumoconiosis and indicated the presence of a large category A opacity in the right upper zone. Dr. Rasmussen stated that the radiographic evidence and the lung biopsy indicated the presence of pneumoconiosis. Dr. Rasmussen explained that the limited amount of surgical material collected through the biopsy prevented the pathologist from providing a definitive diagnosis of complicated pneumoconiosis. However, Dr. Rasmussen believed that there was both x-ray and CT scan evidence which established the presence of complicated pneumoconiosis. (CX 1).

Dr. Rasmussen opined that the ventilatory function studies "revealed moderate, irreversible obstructive ventilatory impairment [and] the single breath carbon monoxide diffusing capacity was very markedly reduced." (CX 1). Dr. Rasmussen had Miner complete an incremental treadmill exercise study. Based upon the results, Dr. Rasmussen concluded that Miner was permanently and totally disabled and not capable of performing any significant gainful employment. He explained that Miner had a "very marked loss of lung function as reflected by his moderate ventilatory impairment, very marked reduction in diffusing capacity and very marked impairment in gas exchange with increased dead space ventilation and marked hypoxia during light exercise." (CX 1).

Dr. Rasmussen opined that both Miner's extensive exposure to coal dust and significant smoking history contributed to Miner's disabling lung disease. He noted that both factors can cause identical forms of COPD and emphysema. Dr. Rasmussen explained that "lung tissue is destroyed by the identical cellular response to inhalation to coal mine dust and cigarette smoke leading to a cascade of cellular and enzymatic processes which destroy lung tissue. Coal mine dust in contrast to cigarette smoke also causes diffuse interstitial fibrosis, which usually accompanies the emphysema." (CX 1). Dr. Rasmussen stated that he evaluated Miner on five separate occasions between 2000 and 2006. Based upon these evaluations, Dr. Rasmussen found that Miner showed "progressive impairment in gas exchange impairment… the pattern of impairment in [Miner's] disabling lung disease is also quite consistent with the effects of coal mine dust exposure including complicated pneumoconiosis or progressive massive fibrosis, (i.e. significant impairment in oxygen transfer in the absence or out of proportion to ventilatory impairment) as seen in this case." (CX 1).

Dr. Rasmussen (DX 14)

Dr. Rasmussen conducted the Department of Labor examination for Miner's current claim and completed a report dated May 10, 2005. Dr. Rasmussen noted that Miner had started smoking irregularly at age 14, started smoking regularly by age 16 and smoked half a pack per day until he quit in 2003. (DX 14). Dr. Rasmussen was informed that Miner had worked in the coal mines for approximately 24.5 years. Dr. Rasmussen reported that Miner had experienced shortness of breath with exertion for approximately fifteen years and was only able to walk slowly on level ground and was limited to climbing less than one flight of stairs. Miner had a minimally productive cough on some mornings, experienced wheezing and coughing at night and in the morning, slept on three pillows and coughed up small amounts of blood on occasion. Dr. Rasmussen noted that Miner had pneumonia in 2000 and a spontaneous pneumothorax in 2004, which lead to a lung biopsy that yielded evidence of pneumoconiosis. (DX 14).

Dr. Rasmussen examined Miner and found his breath sounds to be moderately to markedly reduced. He also found bilateral medium basilar rales, prolonged expiratory phase with forced expirations and faint expiratory wheezes. (DX 14). An x-ray was taken in conjunction with this exam. Dr. Rasmussen reported that the x-ray indicated pneumoconiosis, emphysema and complicated pneumoconiosis, however, with respect to the complicated pneumoconiosis, he mentioned that "other considerations were old granulomata or possible malignancy." (DX 14).

Numerous laboratory studies were also conducted. Dr. Rasmussen concluded that the ventilatory function studies revealed "moderate, partially reversible obstructive ventilatory impairment… and moderate impairment in oxygen transfer at rest." (DX 14). In Dr. Rasmussen's opinion, the exercise studies "indicate very marked loss of lung function as reflected by the patient's very marked reduction in diffusing capacity and impairment in oxygen transfer during very light exercise." Dr. Rasmussen concluded that Miner does not retain the ventilatory or pulmonary capacity to perform his last coal mine job. Dr. Rasmussen also stated that Miner "has a significant history of exposure to coal mine dust. He has x-ray changes

consistent with complicated coal workers' pneumoconiosis. It is medically reasonable to conclude the patient has complicated coal workers' pneumoconiosis, Category A, which arose from his coal mine employment." (DX 14). Dr. Rasmussen opined that both coal mine dust exposure and cigarette smoking contributed to Miner's disabling lung disease. He explained that coal mine dust exposure was a major contributing factor "in view of the pattern of impairment, i.e. only minimal post bronchodilator ventilatory impairment, but yet marked impairment in oxygen transfer during exercise. This is a pattern that is commonly encountered among impaired coal miners." (DX 14).

Hospitalization Records and Treatment Notes

Columbia Clinch Valley Medical Center

Miner was hospitalized at the Columbia Clinch Valley Medical Center between August 5, 2004 through August 16, 2004 in order to receive treatment for pneumothorax and acute shortness of breath. During the hospitalization, Miner received a tube thoracostomy, a fiberoptic bronchoscopy with washings and right thorascopy with biopsy. (EX 4 at 1). The initial pathology report diagnosed bronchoalveolar carcinoma, however, upon further review, it was concluded that the biopsy does not yield a definitive diagnosis of cancer. Miner was maintained with a chest tube. After removal of the tube there was no radiographic evidence of pneumothorax, however there was still evidence of some subcutaneous emphysema. The discharge diagnoses included "pheumothorax, haemophilus influenzae pneumonia, pulmonary fibrosis, alveolar hyperplasia." (EX 4 at1).

Dr. Kadel treated Miner during this hospitalization. Dr. Kadel noted a smoking history of one pack per day for 40 years, a surgical history of endoscopy and a past medical history that included CWP and arthritis. Upon physical examination, Dr. Kadel found Miner's breath sounds to be markedly diminished on the right with no crepitus. Dr. Kadel's impression was "pneumothorax secondary to ruptured emphysematous bleb." (EX 4 at 4). During surgery, Dr. Kadel reported that "there was evidence of dense adhesions of the right upper lobe to the chest wall." He was unable to separate the lung from the chest wall. Following the failed attempt, Dr. Kadel terminated the procedure. (EX 4 at 11).

Dr. Stefanini conducted an evaluation of Miner during his hospitalization at the Clinch Valley Medical Center. Dr. Stefanini noted that 10 hours of severe breathing difficulty prompted Miner's visit to the hospital. Dr. Stefanini reported that Miner had worked in the coal mines for 40 years and had been a heavy smoker. Dr. Stefanini found a "mild decrease of breath sounds on the right with crepitation." He also commented on the finding of a right-sided pneumothorax. "It was felt most likely that this was a pneumothorax secondary to ruptured emphysematous bleb on the basis of patient's chronic pulmonary disease." (EX 4 at 6). Dr. Stefanini opined that "a CAT scan of the chest indicated focal irregular fibrotic area in the mid posterior aspect of the right upper lobe, continuing to the pleural surface with multiple small bullae associated with the density… also minimal chronic interstitial changes were noted in the right base posteriorly and no lymphadenopathy was present." He also reported that the thoracotomy "revealed the presence of tumoral tissue." Upon a limited physical examination, Dr. Stefanini found "much reduction of expansion of both bases… the right lung shows multiple rhonchi and wheezes and percussion

sound is occasionally hyperresonant." (EX 4 at 6). Dr. Stefanini's clinical impressions included "bronchioalveolar carcinoma, upper lobe of right lung, involving pleural surface… chronic obstructive pulmonary disease with emphysematous component and recent pneumothorax…." (EX 4 at 7).

Dr. Titha saw Miner following surgery. He noted a past medical history "suggestive of chronic obstructive pulmonary disease, pneumoconiosis, gastroespohageal reflux disease, osteoporosis, osteoarthritis, anxiety disorder [and] a history of chest pain." He concluded that "pneumothorax has been excellently managed with chest tube by Dr. Kadel." (EX at 8).

Dr. Turjman completed both the cytology report and the biopsy report. The cytology report, signed by Dr. Turjman on August 10, 2004, revealed that the matter obtained in the bronchial washing contained "bronchial epithelial cells, very few, benign neutrophils, many histiocytes; there were no malignant cells..." (EX 4 at 14). Dr. Turjman reviewed a frozen and a fresh section of lung tissue. Based upon the frozen section, Dr. Turjman found "fibrosis of lung tissue with heavy accumulation of carbon pigment with associated extensive chronic inflammatory infiltration [and] localized hyperplasia of alveolar lining cells with secondary inflammatory cellular atypia." (EX 4 at 16). Dr. Turjman reported that the lung tissue in the fresh specimen showed "extensive fibrosis, chronic inflammatory infiltration and carbon pigment accumulation." (EX 4 at 16). Dr. Turjman commented that this was a difficult case; he opined that "there is hyperplasia of alveolar lining cells with cellular atypia… the pattern resembles the pattern of bronchioalveolar carcinoma. However, the cellular proliferation is limited to a small area surrounded by fibrosis with heavy chronic inflammatory infiltration and no distinct nuclear anaplasia is detected on permanent sections." Dr. Turjman did not find malignancy and noted that the Staff at the Department of Pulmonary and Mediastinal Pathology at the Armed Forces Institute of Pathology also failed to find evidence of malignancy in the material.

Miner's fourth exhibit contains seven treatment notes from Dr. Robinette dated from 3/19/04 through 8/29/06. Dr. Robinette was treating Miner for black lung disease and chronic airflow obstruction. (CX 4). In all but the earliest treatment note, Dr. Robinette references the presence of progressive massive fibrosis. (CX 4 at 1,3,4,6,8,10,11 and 14). He based the above diagnosis on radiographic evidence of a category A mass found in the right upper lung. (CX at 1 and 11). In the treatment note dated September 20, 2004, Dr. Robinette stated that Miner had "developed an occupational pneumoconiosis consistent with complicated coal workers' pneumoconiosis and has associated severe COPD with bullous emphysema and status post hemithorax." (CX 4 at 11). Dr. Robinette noted that despite the diagnosis, Miner continued to smoke. (CX 4 at 6 and 8). Dr. Robinette prescribed bronchodilators during the course of treatment, but on August 29, 2006, reported that "[Miner] has been treated with maximum bronchodilators and continues to have cough , congestion and dyspnea." (CX 4 at 1). Throughout these treatment records, Dr. Robinette found that Miner exhibited diminished breath sounds with a prolongation of the expiratory phase. (CX 4 at 1,3, 4, 6, 10 and 14). He also found rhonchi and wheezes on occasion. (CX 1 and 14).

Dr. Robinette commented on biopsy findings from the Staff Pathologist at the Armed Force Institution of Pathology. He noted that the findings were consistent with CWP and "the confluent nature of the fibrotic reaction recorded raises the concern of an underlying diagnosis of progressive massive fibrosis but the biopsy sample made them reluctant to render that diagnosis." (CX 4 at 10). Dr. Robinette reviewed a right thoracotomy specimen and stated that Miner "was found to have anthrasilicotic nodules consistent with CWP and emphysema. There was some cellular atypical present but no diagnosis of malignancy could be identified. There was carbonous pigment present with chronic inflammatory infiltration." (CX 4 at 4).

DISCUSSION

Subsequent Claim Threshold

I must first determine whether the new evidence establishes an element of entitlement that has previously been adjudicated against Miner, namely whether Miner's total disability was caused or contributed to by pneumoconiosis.

New Evidence and Causation

Unless one of the presumptions at 20 C.F.R. §§ 718.304, 718.305, or 718.306 (2000) and (2001) is applicable, a miner must establish that his or her total disability is due, at least in part, to pneumoconiosis. The Board has held that "[i]t is [the] Miner's burden pursuant to § 718.204 to establish total disability due to pneumoconiosis . . . by a preponderance of the evidence." Baumgartner v. Director, OWCP, 9 B.L.R. 1-65, 1-66 (1986); Gee v. Moore & Sons, 9 B.L.R. 1-4, 1-6 (1986) (en banc).

The presumptions at §§ 718.305 and 718.306 are inapplicable because they only apply to claims that were filed before January 1, 1982, and June 30, 1982, respectively. Section 718.304 requires x-ray, biopsy, or equivalent evidence of complicated pneumoconiosis. Complicated pneumoconiosis is diagnosed after a finding of an opacity greater than one centimeter is categorized as a type A, B or C. In this case, there is conflicting evidence pertaining to the existence of complicated pneumoconiosis in Miner's lungs.

In evaluating the x-ray evidence, I assign heightened weight to interpretations of physicians who qualify as either a board-certified radiologist or "B" reader. Dixon v. North Camp Coal Co., 8 BLR 1-344, 1-345 (1985). I assign greatest weight to interpretations of physicians with both of these qualifications. Woodward v. Director, OWCP, 991 F.2d 314, 316 n.4 (6th Cir. 1993); Sheckler v. Clinchfield Coal Co., 7 BLR 1-128, 1-131 (1984). It is proper to accord greater weight to the interpretation of a C-reader over that of a B-reader. Allen v. Riley Hall Coal Co., 6 B.L.R. 1-376 (1983). B-reader's interpretation is entitled to greater weight than that of an A-reader. Pavesi v. Director, OWCP, 758 F.2d 956 (3rd Cir. 1985).

The x-ray evidence does not establish the presence of complicated pneumoconiosis. A board certified radiologist provided a narrative interpretation of the x-ray dated August 29, 2006. He found pulmonary hyperinflation consistent with COPD and nodular interstitial lung disease consistent with CWP. He did not identify an opacity that would qualify as a category A, B, or C opacity. Therefore, I find this x-ray to be equivocal.

Two dually qualified specialists and a B-reader interpreted the May 30, 2006 x-ray. One dually qualified specialist found the x-ray to be negative for both simple and complicated pneumoconiosis. The other dually qualified specialist and the B-reader both interpreted the x-ray as positive for both simple and complicated pneumoconiosis, with a profusion of 1/1 and a type A opacity. I find this x-ray to be positive for complicated pneumoconiosis.

There were four interpretations of three x-rays taken in 2005. The most recent, dated November 24, 2005, was interpreted as positive for simple pneumoconiosis, but negative for complicated pneumoconiosis. The August 2, 2005 x-ray was interpreted by a board certified radiologist and A-reader. He found "interstitial fibrosis, emphysema and some infiltration in the right upper lobe," which he concluded was due to old tuberculosis and therefore his interpretation was negative for complicated pneumoconiosis. The May 10, 2005 x-ray was interpreted by a dually qualified specialist as negative for both simple and complicated pneumoconiosis. A B-reader interpreted this same x-ray as positive for both simple and complicated pneumoconiosis with a profusion of 1/0 and a category "A" opacity. I find all three x-rays taken in 2005 to be negative for complicated pneumoconiosis.

Narrative interpretations were provided for two x-rays requested by Miner's treating physician. Based upon the x-ray dated September 14, 2004, a board certified radiologist noted "an ill defined opacity in the RUL." This comment alone does not establish the existence on an opacity that would be equivalent to a type A, B or C opacity in the lung, therefore I find this x-ray to be equivocal. A board certified radiologist and A-reader interpreted the August 19, 2004 x-ray, but did not find any opacities or mention complicated pneumoconiosis. I find this x-ray to be negative for complicated pneumoconiosis. Numerous x-rays were taken during Miner's hospitalization between August 5, 2004 and August 15, 2004. Most x-rays showed scarring and pleural thickening of the right upper lobe. In the August 16, 2004 x-ray this thickening is "presumed significant fibrosis." However, there is no mention of an opacity that would be greater than one centimeter in size. Many of the x-ray interpretations reference a density in the right subapex. This density was identified as an infiltrate or atelectasis. Improvement in the area of density was seen in the August 6, 2004 x-ray. At no point was the size of this density discussed, nor was this density ever classified as complicated pneumoconiosis. A portable x-ray taken on August 11, 2004 indicated "some linear density now seen in the left lung base consistent with linear atelectasis." The density found in the left lung was not classified as complicated pneumoconiosis. I find that none of the x-rays taken during Miner's hospitalization establish the existence of complicated pneumoconiosis.

In summary, only one of the new x-rays supports the diagnosis of complicated pneumoconiosis. Three of the x-rays were definitively negative for complicated

pneumoconiosis, while the remaining x-rays were equivocal and therefore were of little probative value on the issue. I find that the radiographic evidence does not support a finding of complicated pneumoconiosis under § 718.304.

The biopsy evidence also fails to establish the presence of complicated pneumoconiosis because neither specialist definitely diagnosed complicated pneumoconiosis. Dr. Shilo opined that the biopsy tissue raised the concern of progressive massive fibrosis; however he reported that the limited nature of the biopsy sample made him reluctant to render that diagnosis. Dr. Naeye reviewed the biopsy tissue and stated that there was "no lesion anywhere near the size (2.0 cm) or the microscopic characteristics required to make the diagnosis of complicated coal workers' pneumoconiosis." I find that Miner did not establish complicated pneumoconiosis through biopsy evidence under § 718.304.

Dr. Wiot explained that CT scan evidence may be helpful in diagnosing complicated pneumoconiosis. (EX 1). Dr. Wiot provided an interpretation of the March 26, 2003 CT scan, in which he concluded that there was no evidence of pneumoconiosis. He did report the presence of linear fibrotic stranding with associated pleural disease, but opined that this disease was not a manifestation of coal dust exposure. According to Dr. Hassett, the August 5, 2004 CT scan showed evidence of a density surrounded by several small bullae, but he does not comment on the size. (EX 4). Dr. Begren, McReynolds and Radhakrishnan interpreted the three most recent CT scans. Dr. McReynolds reviewed the April 25, 2005 x-ray and noted a prior finding of progressive massive fibrosis; however he opined that the area in the right upper lobe could either be fibrosis and/ or stable scarring. (CX 4). Based upon the December 2004, CT scan, Dr. Radhakrishnan concluded that Miner had "advanced bullous emphysematous disease with subpleural nodules." (CX 4). He does not comment upon the size of these nodules or discuss the connection between these changes and coal dust exposure. He also mentioned "interval progression of scarring in the right apex." (CX 4). He does not identify an opacity in the right lung.

Dr. Bergren is the only doctor to definitively diagnose progressive massive fibrosis. Dr. Bergren interpreted the September 1, 2005 CT scan and reported that there were "stable fibrotic changes in the right greater than left upper lobes consistent with progressive massive fibrosis." (CX 4). Dr. Bergren does not provide enough information for me to determine whether the changes he identified would be equivalent to the radiographic finding of a type A, B or C opacity. Considering the fact that Dr. Bergren is the only physician that definitively diagnosed progressive massive fibrosis and his diagnosis was not clear enough for me to make an equivalency finding, I find that the CT scan evidence does not support a finding of complicated pneumoconiosis.

Miner was unable to establish the presence of complicated pneumoconiosis through x-ray, biopsy or CT scan evidence. There is no other new evidence that would either refute or support a finding of complicated pneumoconiosis. I find that the presumption found at 20 C.F.R. §§ 718.304 is not applicable.

The amended regulations at 20 C.F.R. §718.204(c) (2001) contain a standard for determining whether total disability is caused by the miner's pneumoconiosis and provides the following:

> (c)(1) Total disability due to pneumoconiosis defined. A miner shall be considered totally disabled due to pneumoconiosis if pneumoconiosis, as defined in Sec. 718.201, is a *substantially contributing cause* of the miner's totally disabling respiratory or pulmonary impairment. Pneumoconiosis is a 'substantially contributing cause" of the miner's disability if it: (i) Has a material adverse effect on the miner's respiratory or pulmonary condition; or (ii) Materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment.
>
> (2) Except as provided in Sec. 718.305 and paragraph (b)(2)(iii) of this section, proof that the miner suffers or suffered from a totally disabling respiratory or pulmonary impairment as defined in paragraphs (b)(2)(i), (b)(2)(ii), (b)(2)(iv) and (d) of this section shall not, by itself, be sufficient to establish that the miner's impairment is or was due to pneumoconiosis. Except as provided in paragraph (d), the cause or causes of a miner's total disability shall be established by means of a physician's documented and reasoned medical report.

20 C.F.R. §718.204(c) (2001) (emphasis added). [23]

A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. Fields v. Island Creek Coal Co., 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. Hoffman v. B&G Construction Co., 8 B.L.R. 1-65 (1985); Hess v. Clinchfield Coal Co., 7 B.L.R. 1-295 (1984). A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. Fields, supra. Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder-of-fact to decide. Clark v. Karst-Robbins Coal Co., 12 B.L.R. 1-149 (1989)(en banc).

Dr. Rasmussen provided the only two medical reports offered as new evidence. Each report was based upon a physical examination of Miner, an x-ray, a blood gas study, a pulmonary function test, a review of Miner's symptoms and Miner's family, occupational and social histories. The most recent report also contained Dr. Rasmussen's review of the biopsy evidence. I find this data to be sufficient to yield a well-documented opinion.[24] In both reports

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[23] In its comments, the Department noted that addition of the word "material" or "materially" to the foregoing provisions reflects the view that "evidence that pneumoconiosis makes only a negligible, inconsequential, or insignificant contribution to the miner's total disability is insufficient to establish that pneumoconiosis is a substantially contributing cause to that disability." Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969, 65 Fed. Reg. 79,946 (Dec. 20, 2000).

[24] In Harris v. Old Ben Coal Co., 23 B.L.R. 1-98 (2006)(en banc)(J. McGranery and J. Hall, concurring and dissenting), the Benefits Review Board indicated that when confronted with a medical opinion that contained

Dr. Rasmussen diagnosed Miner with pneumoconiosis, found Miner to be totally disabled and opined that both pneumoconiosis and tobacco abuse contributed to Miner's disabling lung disease. Although Dr. Rasmussen asserted that cigarette smoking and coal dust exposure can cause identical forms of COPD and emphysema, he opined that the "significant impairment in oxygen transfer in the absence or out of proportion to ventilatory impairment," and the minimal post bronchodilator ventilatory impairment paired with marked impairment in oxygen transfer during exercise exhibited by Miner was consistent with the effects of coal dust exposure." Therefore, Dr. Rasmussen provided an explanation of how the underlying data supported his conclusions. I find his explanation to be credible, and therefore I find both of his reports to be well-reasoned.

Although Dr. Rasmussen did not specify the proportion of impairment caused by pneumoconiosis and tobacco, I still find his opinion to be probative on the issue. In Tapley v. Bethenergy Mines, Inc., BRB No. 04-0790 BLA (May 26, 2005) (unpub.), the ALJ properly found that a physician's opinion that coal workers' pneumoconiosis constituted one of two causes of Miner's totally disabling respiratory impairment satisfied the causation standard at 20 C.F.R. § 718.204(c)(1). Citing to Gross v. Dominion Coal Corp., 23 B.L.R. 1-8, 1-17 to 1-19 (2004), the Board noted that a medical opinion that pneumoconiosis "was one of two causes" of the miner's total disability met the "substantially contributing cause" standard. See also Consolidation Coal Co. v. Director, OWCP [Williams], 453 F.3d 609 (4th Cir. 2006). (the Court held that physicians need not "apportion [Miner's] lung impairment between cigarette smoke and coal mine dust exposure…" in order to provide probative evidence concerning the cause of Miner's total disability).

In each report Dr. Rasmussen based his conclusions on a smoking history that is not consistent with the finding of this Court. In the first report, Dr. Rasmussen based his conclusions on a 20 pack year smoking history, whereas in the most recent report, Dr. Rasmussen notes a smoking history of between 40 and 50 pack years. I found that Miner had a 32 pack year smoking history. In Trumbo v. Reading Anthracite Co., 17 B.L.R. 1-85 (1993), the Board held that a physician's opinion may be less probative if based upon an inaccurate smoking history. A twenty pack year smoking history, while still significant, is substantially less than a 32 pack year history. I grant Dr. Rasmussen's first report less weight. The most recent report was based upon a smoking history of between 40 and 50 pack years, between 8 and 18 pack years greater than the finding of this Court, yet Dr. Rasmussen still opined that pneumoconiosis had an affect on Miner's disabling lung impairment. I therefore find that Dr. Rasmussen's inflated estimation of Miner's smoking history has no bearing on the credibility of his most recent opinion.

Miner had a number of treating physicians, however the treatment records pertinent to the subsequent claim threshold do not contain an adequate explanation of the etiology of his impairment.

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evidence not admitted into the formal record, an administrative law judge may: a) exclude the report; b) redact the objectionable content; c) require a revised report; or, d) consider the physician's reliance on the inadmissible evidence in deciding the probative value of the report. I did not admit the pulmonary function study taken in conjunction with Dr. Rasmussen's 2006 report. Dr. Rasmussen's opinion was based primarily upon the arterial blood gas studies, and he had access to the pulmonary function test results taken in conjunction with prior exams. Therefore, I still find Dr. Rasmussen's opinion to be probative.

Miner has offered two well-documented medical opinions regarding the etiology of his disabling lung disease. Although the first report is less credible due to the reliance on an inaccurate smoking history, the most recent report confirms Dr. Rasmussen's earlier conclusions. Employer did not offer new evidence to rebut these two reports. I therefore find Miner has established that pneumoconiosis contributed to his disabling pulmonary impairment under 20 C.F.R. §718.204(c) (2001). Accordingly, Miner has proven an element of entitlement that was previously adjudicated against him. I must therefore open the entire record in order to determine Miner's eligibility for Black Lung Benefits.

De Novo Review of the Record

Now that the record has been reopened and this decision is based upon de novo review and consideration of the administrative record as a whole, not all of the evidence that has been introduced prior to the instant subsequent claim will be listed except as required for an analysis of entitlement.[25]

Dr. James R. Castle *(DX 3, folder 3, formerly EX 3)*

Dr. Castle evaluated the Miner on March 26, 2003. (DX 3 formerly EX 3). The Miner reported trouble with shortness of breath for at least the preceding 15 years and also indicated heart trouble, although the cause had not been identified. Dr. Castle noted that Miner smoked half a pack per day for nearly 40 years, yielding an approximate 20 pack year smoking history. Dr. Castle based his conclusions upon a reported occupational history of over twenty four years of coal mine employment, ending in 1991.

On examination of the Miner, Dr. Castle noted coarse rhonchi bilaterally, which cleared with coughing. The Miner had no rales, crackles, crepitations, or wheezes. The Miner's chest x-ray was positive for pneumoconiosis with a profusion of 1/1. There were no large opacities. There was also evidence of significant bullous emphysema and calcified granulomas, which was supported by the findings from the March 26, 2003 CT scan. The CT scan also showed some nodules that were not calcified. In Dr. Castle's opinion, these changes were most likely indicative of granulomatous disease, and not related to coal workers' pneumoconiosis. He conceded that it was possible that a few of

[25] I have reviewed the medical evidence associated with the prior claims and I did not find any discrepancies between the medical evidence and the summaries of the evidence provided by Judge Chapman or Judge Wood. Because pneumoconiosis is a progressive and irreversible disease, it may be appropriate to accord greater weight to the more recent evidence of record, especially where a significant amount of time separates newer evidence from that evidence which is older. Clark v. Karst-;Robbins Coal Co., 12 B.L.R. 1-;149 (1989)(en banc); Casella v. Kaiser Steel Corp., 9 B.L.R. 1-;131 (1986). I find the medical evidence associated with the proceeding before Judge Wood to be of little probative value in this analysis considering the significant amount of time that separates that evidence from the newer evidence associated with the instant subsequent claim and the claim before Judge Chapman. I therefore do not summarize the medical evidence associated with the proceeding before Judge Wood. I do adopt Judge Chapman's summarization of the medical evidence, which I have modified or condensed when necessary.

the noncalcified nodules could be related to pneumoconiosis, but in his opinion, the overall changes are most likely due to granulomatous disease.

Pulmonary function studies and arterial blood gas studies were conducted during Dr. Castle's examination. The Miner's pulmonary function studies, which Dr. Castle found to be valid, showed evidence of moderate airway obstruction, without change after bronchodilators. The lung volume studies showed hyperinflation and gas trapping. The Miner's diffusing capacity was severely reduced, but his DL/VA was moderately reduced. The Miner's arterial blood gas study showed evidence of normal oxygenation, with very marked hyperventilation. The carboxyhemoglobin level was normal.

In Dr. Castle's opinion, there was insufficient objective medical data collected during his examination of Miner and found in Miner's medical records to indicate the presence of pneumoconiosis. He concluded that the Miner most likely did not have coal workers' pneumoconiosis, and that his x-ray changes were due to granulomatous disease. Dr. Castle opined that Miner had tobacco smoke induced bullous emphysema, and moderate airway obstruction with gas trapping, hyperinflation, and reduction in diffusing capacity due to tobacco smoke induced bullous emphysema. He had pulmonary emphysema due to his long and extensive history of tobacco abuse.

Although Dr. Castle felt that the Miner worked in or around the underground mines long enough to have developed pneumoconiosis if he were a susceptible host, he also noted that Miner's smoking history was a risk factor for the development of pulmonary symptoms. According to Dr. Castle, this history is sufficient to have caused the Miner to develop chronic obstructive pulmonary disease, if he were a susceptible host. Dr. Castle noted that heart disease is also another risk factor for the development of pulmonary symptoms. The Miner was told that he had heart disease, although he was not clear on the type.

According to Dr. Castle, the Miner never demonstrated consistent physical findings indicating the presence of an interstitial pulmonary process, and he did not have consistent findings of rales, crackles, or crepitations.

Dr. Castle opined that the valid physiologic studies done most recently showed evidence of moderate airway obstruction, without significant change after bronchodilators. There was also evidence of hyperinflation, gas trapping, and reduction in diffusion capacity. Dr. Castle considered these findings to be inconsistent with changes due to pneumoconiosis, but rather indicative of tobacco smoke induced bullous emphysema. Miner did not exhibit any degree of restriction and Dr. Castle explained that when pneumoconiosis causes impairment, it results in a mixed, irreversible obstructive and restrictive ventilatory defect, which was not the finding here. Dr. Castle noted progression since his examination of Miner in 1995, at which point Miner only exhibited

mild airway obstruction. Dr. Castle attributes this progression to changes caused by bullous emphysema, rather than due to any form of pneumoconiosis.

Dr. Castle found it significant that the Miner had considerable variability in study values.[26] In Dr. Castle's opinion this variability indicated some degree of reversibility in the chronic airway obstruction, a finding that is indicative of tobacco smoke induced airway obstruction, as opposed to obstruction associated with pneumoconiosis. Dr. Castle explained that pneumoconiosis does not cause a reversible airway obstruction. Additionally, the significant reduction in diffusion capacity associated with hyperinflation and gas trapping was indicative of tobacco smoke induced bullous emphysema. According to Dr. Castle, pneumoconiosis does not typically cause a reduction of diffusion capacity, but when it does, it is in the presence of a high degree of profusion of p/r type opacities, which was not the finding here. Dr. Castle also believed that the Miner's hypoxemia was related to his tobacco smoke induced pulmonary emphysema.

In Dr. Castle's opinion, Miner was permanently and totally disabled due to tobacco smoke induced bullous emphysema. However, his disability was not consistent with any pulmonary process that has arisen from his coal mining employment. Miner had evidence of at least moderate airway obstruction, with some degree of reversibility, and a significant reduction in diffusion capacity, associated with hyperinflation and gas trapping. Such findings are totally indicative of tobacco smoke induced bullous emphysema, and not related in any way to the inhalation of coal mine dust or coal workers' pneumoconiosis. Dr. Castle testified in his May 12, 2003 deposition that lung volumes in pulmonary emphysema are markedly increased, or hyperinflated, with gas trapped in the lung because of airway collapse. But the fibrotic process in coal workers' pneumoconiosis is the exact opposite, and causes shrinkage of the lung; even though there may be some airway obstruction, one would expect the lung volumes to be on the lower side.

In his deposition, dated May 12, 2003, (DX 3 formerly EX 5) Dr. Castle explained the difference between changes caused by bullous emphysema and changes related to coal mine dust exposure. He stated that bullous emphysema is not related to coal mine dust exposure; it is seen with tobacco smoking, and a congenital condition called alpha-1 antiprotease deficiency. According to Dr. Castle, the emphysema that is a part of pneumoconiosis is defined as focal emphysema, which is seen as part of the coal macule microscopically. It is not large enough to be seen on x-ray or with the naked eye. Nor does bullous emphysema cause the development of a large solid nodule.

[26] When he was examined by Dr. Fino in October 2000, the FEV1 was 56% of predicted, compared to the 71% of predicted in Dr. Castle's most recent examination. At Dr. Rasmussen's September 2000 examination, the FEV1 was 57% of predicted.

In Dr. Castle's opinion the apparent worsening of the Miner's condition since 1995 is related to the progression of the tobacco smoke induced bullous emphysema. According to Dr. Castle, the records clearly showed that the Miner continued to smoke long after he left the mines, resulting in the progression of his tobacco smoke induced lung disease.

Dr. Castle stated that his opinion about the lack of disability due to that process would remain the same even if he had concluded that Miner had radiographic evidence of pneumoconiosis. He noted that this opinion was not predicated on the x-ray, but relied on the fact that Miner did not have the physiologic abnormalities that indicate disability due to pneumoconiosis.

Dr. Emory Robinette (DX 3, folder 3 formerly CX 7)

While Dr. Robinette did not specifically comment on the etiology of Miner's disability in the treatment notes pertinent to the subsequent claim threshold, he did comment upon the etiology in a deposition for the claim before Judge Chapman. (DX 3 formerly CX 7). Dr. Robinette testified on July 3, 2003 that he had treated Miner from 1999 through 2002. (DX 3 formerly CX 7) Dr. Robinette diagnosed Miner with COPD and underlying pneumoconiosis. He also concluded that Miner was totally impaired. Dr. Robinette acknowledged that cigarette smoking is a significant cause of COPD, but he also stated that there was a direct correlation between coal dust deposition and the development of emphysematous change. He testified that it is recognized that coal miners who smoke cigarettes develop much more progressive disease, and more debilitating disease than individuals who smoked but did not work in the coal mines. In general, working one year as an underground miner is equal to smoking about three quarters of a pack of cigarettes a day, in commonly cited references. There is no way to say that one factor caused most of the abnormalities or disabilities, but in this particular case, one could not say that the coal mining did not cause any of it.

Dr. Robinette stated that the Miner's history of dust exposure, his x-rays, and the ausculatory findings are consistent with intrinsic lung disease in individuals with pneumoconiosis, and is associated with emphysematous changes. He noted that typically, smokers he treats for severe reduction in diffusion capacity also have severe reduction of FEV1 and FVC or airflow, but not just selective impairment of diffusion capacity and mild airflow obstruction.

Dr. Robinette concluded that Miner had a significant amount of emphysema associated with coal dust deposition, and a large amount of obstruction, which results in the testing abnormalities and his profound breathlessness. He disagreed with Dr. Castle that pneumoconiosis does not typically cause a reduction in diffusion capacity. Dr. Robinette stated that any disease process that can cause emphysematous change can cause a reduction in the diffusion capacity, as clearly documented in the literature. He

explained that a significant reduction in diffusion capacity was typically seen in emphysema, and in his opinion, the Miner's pneumoconiosis certainly contributed to his emphysema. Dr. Robinette opined that Miner's pulmonary function and arterial blood gas studies have not been variable; the airflow pattern is consistent. His pulmonary function studies do not show significant reversibility. (DX 3 formerly CX 7).

Dr. D.L. Rasmussen (DX 3, folder 2, formerly DX 13)

Dr. Rasmussen examined the Miner at the request of the Department of Labor on September 25, 2000. (DX 3 formerly DX 13). He noted an occupational history of 24.5 years of coal mine employment and reported that the Miner smoked half to one pack per day from 1963 through 1991. On examination of the Miner, Dr. Rasmussen noted moderately reduced breath sounds on auscultation, but no rales, rhonchi, or wheezes. There was a prolonged expiratory phase with forced respiration.

The Miner's x-ray showed pneumoconiosis with a profusion of 2/2, with a right axillary nodule that was most likely a granuloma. His pulmonary function study showed a moderate, irreversible obstructive ventilatory impairment, and his arterial blood gas study showed a marked impairment in oxygen transfer during very light exercise. Dr. Rasmussen's diagnosis of coal workers' pneumoconiosis was based on Miner's 24 ½ years of coal mine employment, and the x-ray changes of pneumoconiosis; and COPD/emphysema was based on the airflow obstruction and reduced single breath diffusing capacity for carbon monoxide. In his opinion, the Miner's pneumoconiosis was due to his coal dust exposure, and his COPD/emphysema was due to a combination of his coal mine dust exposure and cigarette smoking. Dr. Rasmussen concluded that Miner had severe loss of lung function, and was totally disabled from performing his last coal mine job, or significant gainful employment. He felt Miner's two risk factors contributing to his impairment were his cigarette smoking and his coal mine dust exposure, with the latter being a significant contributing factor.

Dr. Gregory J. Fino (DX ,3 folder 2, formerly DX 31; DX 3, folder 3, formerly EX 6)

Dr. Fino examined Miner on October 30, 2000, and reviewed Miner's medical records; he also testified by deposition on May 15, 2003. (DX 3 formerly DX 31; DX 3 formerly EX 6). He noted that the Miner worked 25 years in the underground mines, and smoked from one half to one full pack per day for about 22 years, ending in 1985. Dr. Fino also noted that other smoking histories showed that he was still smoking as of 2002, up to two to three packages of cigarettes a day. According to Dr. Fino, this is an adequate exposure history to cause a respiratory impairment in a susceptible host, as was his exposure to coal mine dust.

Dr. Fino interpreted Miner's x-rays as negative for pneumoconiosis, reviewed other chest x-rays and concluded that there was no radiographic evidence of pneumoconiosis. He saw no rounded opacities in the upper lung zones; there was old granulomatous disease relating to a previous infection in the right upper lung field. Dr. Fino did find radiographic evidence of bullous emphysema, or large airway destruction which can be seen with the naked eye. The CT scan evidence also supported the finding of emphysema. He stated that bullous emphysema is not caused by exposure to coal mine dust; it is either due to smoking, or it is hereditary.

Dr. Fino noted that the Miner's pulmonary function tests show an obstructive ventilatory abnormality, based on the reduction in the FEV1/FVC ratio. Miner's obstructive ventilatory abnormality occurred in the absence of any interstitial abnormality, and showed involvement in the small airways. According to Dr. Fino, the fact that the small airway flow was more reduced than the large airway flow was not consistent with a coal dust related condition, but is consistent with cigarette smoking, pulmonary emphysema, non-occupational chronic bronchitis, and asthma.

Dr. Fino explained that minimal obstructive lung disease has been described in working coal miners, called industrial bronchitis, which resolves within six months of leaving the mines. He stated that obstructive lung disease can also arise from coal workers' pneumoconiosis, if there is significant fibrosis, which results in the obstruction. Here, Miner's obstruction was unrelated to coal mine dust exposure. Dr. Fino also noted that the total lung capacity was not reduced, ruling out the presence of restrictive lung disease and significant pulmonary fibrosis.

Dr. Fino stated that the Miner's pulmonary function study results were consistent with chronic obstructive bronchitis and emphysema. He also had mild hypoxemia consistent with lung disease. According to Dr. Fino, Miner had over-inflated lungs, reflected by increased lung volumes, and a reduction in diffusion capacity consistent with emphysematous lung destruction. This is a pattern typical in chronic obstructive bronchitis and emphysema. He felt that the studies and examinations both before and after were consistent with his conclusions. Although the Miner has a fixed obstruction, the improvement in pulmonary function studies during Dr. Robinette's and Dr. Castle's examinations showed variability in function, which is not consistent with pneumoconiosis because pneumoconiosis does not improve over time. (DX 3 formerly DX 31; DX 3 formerly EX 6).

According to Dr. Fino, pneumoconiosis can be progressive. (DX 3 formerly EX 6). However, Miner's x- ray did not show changes consistent with such a progression, nor was there a progressive decrease in his lung volumes. His obstruction progressed, which was due to his continued smoking. He felt that Miner's elevated gas trapping and reduced diffusion capacity were consistent with emphysema, and he ruled out scarring or pulmonary fibrosis due to classical pneumoconiosis. He found no indication of pneumoconiosis, either by

physical examination or objective testing. In Dr. Fino's opinion, Miner was totally disabled from a pulmonary standpoint, but his coal dust exposure did not contribute to that impairment. (DX 3 formerly DX 31).

In his report, Dr. Fino discussed pneumoconiosis and its progression at length, citing to numerous studies. He felt that the answer to the question of why some miners had progression of their simple pneumoconiosis and others did not lay in the proper definition of pneumoconiosis. He felt that it was important to differentiate simple coal workers' pneumoconiosis and silicosis; miners whose conditions progressed over time after exposure ceased were miners who had likely contracted silicosis, not pneumoconiosis. He noted that mining jobs that put a worker at risk of silicosis included drilling and roof bolting. (DX 3 formerly DX 31).

Dr. Fino stated **"**Silicosis may be a progressive disease in a small percentage of miners after coal mine dust exposure ends. The literature does not support the statement that coal workers' pneumoconiosis is progressive absent further exposure." According to Dr. Fino, there are no studies that show progressive impairment in miners who have left the mines. (DX 3 formerly DX 31).

Dr. Fino also discussed studies dealing with coal mine dust and obstruction, stating that it was possible to differentiate between the obstruction caused by coal mine dust, and the obstruction caused by other factors such as smoking and asthma. According to Dr. Fino, the studies show that while there can be statistically significant obstruction in some miners, they do not show that the obstruction is clinically significant. The literature does not support a conclusion that coal mine dust inhalation causes a clinically significant reduction in FEV1. Dr. Fino proceeded to critique a number of studies that showed obstruction in coal miners, for bias and other limitations.

Dr. Fino stated **"**There is no doubt that some miners do have clinically significant obstruction as a result of coal mine dust inhalation. This actually is expected in most cases of severe fibrosis where a combined obstructive and restrictive defect is present. However, there is no evidence that there is a clinically significant reduction in the FEV1 as a result of chronic obstructive lung disease due to coal mine dust inhalation."
Dr. Fino's conclusion, after reviewing the studies, was that coal mine dust may cause slight, clinically insignificant decreases in the FEV1 in some miners, but there is no credible scientific support for the proposition that the type of emphysema that arises from dust exposure is impairing or disabling, in the absence of progressive massive fibrosis. (DX 3 formerly DX 31; DX 3 formerly EX 6).

Dr. John A. Michos (DX 3, folder 2, formerly DX 37)

Dr. Michos reviewed medical records at the request of the Director, and provided a report dated February 22, 2001 (DX 3 formerly DX 37). He concluded that the Miner does not have evidence of simple pneumoconiosis, nor a total respiratory disability due to simple pneumoconiosis. He based this conclusion on the fact that the majority of x-ray interpretations were negative, as well as the Miner's significant history of tobacco abuse.

He noted that, while typically simple pneumoconiosis is not associated with a significant decline in ventilatory status, significant tobacco abuse is well known to cause a decline in lung function as seen in the Miner. He felt that if the Miner were found to have pneumoconiosis with a profusion of 2/2 or greater, he could not exclude simple pneumoconiosis as a factor in the Miner's decline in lung functions

Etiology of Total Disability

In addition to Dr. Rasmussen's more recent reports, I find the opinions of Dr. Castle, Dr. Robinette, Dr. Michos and Dr. Fino to be relevant to the analysis of whether Miner's pneumoconiosis caused or contributed to his totally disabling lung disease.

In his report Dr. Fino distinguishes between simple coal workers' pneumoconiosis and silicosis caused by coal dust exposure. Dr. Fino stated that, while silicosis can be progressive, pneumoconiosis is not. In the regulations, both simple coal workers' pneumoconiosis and silicosis caused by exposure to coal mine dust are included under the definition of clinical pneumoconiosis. 20 CFR § 718.201(a)(1). And the regulations provide that "for purposes of this definition, 'pneumoconiosis' is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure." 20 CFR § 718.201(c). I find Dr. Fino's opinions to be inconsistent with the definitions provided in the regulations and therefore grant him less weight.[27] Also Dr. Fino does not believe that disabling obstructive disease can be caused by pneumoconiosis. Finally, Dr. Fino relied on the fact that the Miner's obstruction occurred with a lack of any interstitial abnormality, when in fact several of the x-ray interpretations have noted the presence of interstitial fibrosis. Thus, I do not rely on Dr. Fino's opinions.

Dr. Michos concluded that the Miner does not have evidence of simple pneumoconiosis, nor a total respiratory disability due to simple pneumoconiosis. Dr. Michos made the statement that Miner's impairment could not be caused by pneumoconiosis without radiographic evidence of pneumoconiosis with a profusion of 2/2 or greater. He does not provide any support or explanation of this statement. I find Dr. Michos' opinion to be conclusory, and therefore of little probative value.

Dr. Castle concluded that pneumoconiosis neither caused nor contributed to Miner's totally disabling pulmonary impairment. Dr. Castle based his conclusions on the review of Miner's medical records and a physical examination of Miner, which included

[27] The Board has held that the administrative law judge may discredit the opinion of a physician whose medical assumptions are contrary to, or in conflict with, the spirit and purposes of the Act. Wetherill v. Green Construction Co., 5 B.L.R. 1-248, 1-252 (1982). Although the holdings of the Seventh Circuit are not controlling in this case, I do find their holding in Roberts & Schaefer Co. v. Director, OWCP [Williams], 400 F.3d 902 (7th Cir. 2005) to be persuasive. In Roberts the Seventh Circuit granted less weight to a physician's opinion that contrary was to the provisions at 20 C.F.R. § 718.201(c) providing that pneumoconiosis can be latent and progressive.

an x-ray, a pulmonary function test and an arterial blood gas study. I find this data to be sufficient and therefore find Dr. Castle's examination to be well-documented.

Although Dr. Castle stated that there was insufficient evidence to support a finding of pneumoconiosis, he did clarify that his opinion regarding the etiology of Miner's impairment would remain unchanged even if Miner were found to suffer from the disease.[28] Dr. Castle based his opinion regarding the etiology of Miner's disability primarily on the lack of physical and physiological findings consistent with pneumoconiosis. During his exam, Miner did not exhibit rales, crackles or crepitations, which are findings consistent with pneumoconiosis. Dr. Castle focused mainly on the physiologic studies. Dr. Castle opined that the findings, namely moderate airway obstruction, with some degree of reversibility and a significant reduction in diffusion capacity, were totally indicative of tobacco smoke induced bullous emphysema. Dr. Castle explained that pneumoconiosis was not reversible, and therefore the noted reversibility was not consistent with a finding of pneumoconiosis. He also explained that smoking and coal dust exposure generally caused two different forms of emphysema; coal dust exposure caused focal emphysema whereas smoking caused bullous emphysema. According to Dr. Castle, Miner had bullous emphysema and all of the physiologic findings were related to this form of emphysema. Dr. Castle effectively supported his conclusions with the underlying documentation, and I therefore find his opinion to be well-reasoned.

Drs. Robinette concluded that pneumoconiosis contributed to Miner's totally disabling pulmonary impairment. Dr. Robinette stated that there is a direct correlation between coal dust deposition and the development of emphysematous change. Dr. Robinette opined that any disease process that can cause emphysematous change can cause a reduction in the diffusion capacity. He stated that the reduction in diffusion capacity in this particular case indicated the presence of emphysema and he concluded that pneumoconiosis contributed to this emphysema. Dr. Robinette testified that the smokers he treated typically had severe reduction in diffusion capacity paired with reduction of FEV1 and FVC or airflow. Miner had only selective impairment of diffusion capacity and mild air flow obstruction. While this testimony may be interpreted to mean that the impairment is not likely caused by cigarette smoking, it does not explain

[28] The Board held in Scott v. Mason Coal Co., 289 F.3d 263 (4th Cir. 2002) that it is proper to discredit the opinions of physicians regarding the cause of disability where they concluded that the miner did not suffer from coal workers' pneumoconiosis contrary to the administrative law judge's findings. Some of these physicians opine that, even if coal workers' pneumoconiosis was established, their opinions as to the cause of the miner's total disability would not change. Even with this premise, their reports are not well-reasoned and carry little probative value. See Scott, supra. Dr. Castle went further than simply stating that his opinions would not change. He entertained the possibility of the presence of pneumoconiosis and conceded that certain nodules may in fact be caused by pneumoconiosis. Also, regardless of the failure to find pneumoconiosis, Dr. Castle provided a very thorough opinion of why Miner's impairment is not consistent with pneumoconiosis nor a contributing cause of Miner's impairment.

how these physiologic results indicate that *pneumoconiosis* significantly contributed to the impairment. He also noted that Miner's pulmonary function studies do not show significant reversibility. Pneumoconiosis yields an irreversible impairment, however Dr. Robinette did not specify whether smoking could also yield an irreversible impairment. Therefore, noting that Miner's pulmonary function studies did not indicate any reversibility does not refute the contention that smoking is the sole cause of the impairment.

Dr. Robinette opined that there was no way to determine what proportion of the impairment was caused by cigarette smoking and pneumoconiosis, but he concluded that "one could not say that the coal mining did not cause any of it." He based the above statement on the contention that it is recognized that individuals who smoked but did not work in the coal mines developed less debilitating disease than miners who smoked. He also reported that commonly cited references state that one year of work underground in a mine is equal to smoking approximately ¾ of a pack of cigarettes a day for a year. These statements may refer to a documented trend amongst smokers, however they do not offer proof as to whether this trend is manifested in this particular case. Nor do these statements rely upon the medical data acquired in this claim. Dr. Robinette failed to support his conclusions with the documentation related to this claim, therefore I do not find Dr. Robinettes's opinion to be well-reasoned.

Standing alone, Dr. Rasmussen's opinion was sufficient to allow me to find that Miner's impairment was contributed to by his pneumoconiosis during the subsequent claim threshold analysis. However, Dr. Castle's report effectively rebutted Dr. Rasmussen's conclusions. Dr. Rasmussen's report is the more recent report. However, I do not find this to be determinative of the issue because Dr. Rasmussen's findings related to each of his more recent examinations are in line with Dr. Castle's findings.[29]

First Dr. Castle recognized that Miner had a severe pulmonary impairment. He acknowledged Miner's extensive coal mine employment and that coal dust exposure could cause totally disabling pneumoconiosis; he still concluded that this impairment was completely due to bullous emphysema. He noted that the form of emphysema exhibited by Miner is typically caused by cigarette smoking and not pneumoconiosis. Dr. Castle reviewed Miner's medical records and previous exams and identified patterns of impairment, which he used to further support his contention that Miner's impairment was not contributed to by pneumoconiosis. He then used the clinical data to explain why the manifestation of the impairment could not be caused by pneumoconiosis.

[29] A medical report containing the most recent physical examination of the miner ***may*** be properly accorded greater weight as it is likely to contain a more accurate evaluation of the miner's current condition. Gillespie v. Badger Coal Co., 7 B.L.R. 1-839 (1985). See also Bates v. Director, OWCP, 7 B.L.R. 1-113 (1984) (more recent report of record entitled to more weight than reports dated eight years earlier); Kendrick v. Kentland-Elkhorn Coal Co., 5 B.L.R. 1-730 (1983). I did consider the fact that Dr. Rasmussen's physical examination is more recent, but I do not find that fact to be of any substantial consequence in my analysis of this particular case.

Dr. Castle opined that cigarette smoking and coal dust exposure cause two different forms of emphysema. He explained that cigarette smoking causes bullous emphysema, while pneumoconiosis causes focal emphysema. Dr. Castle reported that Miner suffered from severe bullous emphysema, which is still supported by the more recent treatment records. Dr. Rasmussen's response to this contention was that a significant smoking history and extensive coal mine dust exposure "can cause chronic obstructive lung disease including emphysema. In fact, they cause identical forms of COPD and emphysema including centriacinar, panacinar, bullous, etc." He went on to explain that both factors can cause identical "cellular and enzymatic processes which destroy lung tissue… coal mine dust in contrast to cigarette smoke also causes diffuse interstitial fibrosis, which usually accompanies the emphysema." I find Dr. Castle's opinion to be more persuasive based on his superior qualifications as a board certified pulmonary specialist.

Dr. Rasmussen opined that Miner exhibited a progressive impairment. He stated that "[Miner's] coal mine dust exposure is a significant contributing factor. He has had progressive impairment in lung function." (CX 1). He offered numerous manifestations of this progressive impairment, but he fails to give any explanation of the cause. He did not discuss the tendency for pneumoconiosis to cause a progressive impairment, nor does he provide a reason why tobacco smoke would not be the sole contributor to the progression of impairment. Dr. Castle also noted a progressive impairment in his 2003 report. He attributed this progression to cigarette smoke induced bullous emphysema and explained that while pneumoconiosis could be progressive, that the progression was much more inline with bullous emphysema especially considering Miner's chronic tobacco abuse.

While the presence of severe bullous emphysema, a substantial smoking history, the possibility of a reversible impairment merely indicate that Miner's impairment is not likely contributed to by pneumoconiosis, these findings do not exclude the possibility of pneumoconiosis as a contributing factor. However, Dr. Castle did use the clinical data to support a contention that would rule out pneumoconiosis as a substantially contributing factor. Dr. Castle opined that pneumoconiosis does not typically cause a reduction in diffusion capacity, but when it does, it is in the presence of a high degree of profusion of p/r type opacities. Dr. Rasmussen recently reported that Miner still exhibited a *marked* reduction in diffusion capacity; however the highest profusion identified in any of the recent chest x-rays is 1/1. I therefore find that Dr. Castle's statement is still relevant and credible.

Dr. Castle is also more qualified than Dr. Rasmussen. Dr. Castle practiced with the Pulmonary Medicine Associated since 1977, and at the time of Dr. Castle's report, he was a principal investigator for clinical study trials at Pulmonary Occupational and Research Consultants. He is a member of various societies including the American College of Physicians.

He has published numerous articles and given numerous presentations on topics such as COPD, ARDS, Pneumonia, smoking cessation, etc. (DX 3, former EX 4). Dr. Rasmussen's current CV is not included in record.[30] In Dr. Rasmussen's 2005 report, he does cite to a journal article that he wrote pertaining to the impairment of oxygen in non-smoking coal miners. (CX 1). Unlike Dr. Castle, there is a reference to one of Dr. Rasmussen's articles that specifically pertains to the population in question, namely coal miners. Both physicians are B-readers and board certified in internal medicine. [31] However, Dr. Castle is also board certified in pulmonary disease, has conducted research and given presentations about pulmonary impairment.

Had Dr. Rasmussen properly and sufficiently addressed the trends noted by Dr. Castle, I might have found that Dr. Rasmussen's opinion would be sufficient to prove that pneumoconiosis had a material adverse effect on miner's respiratory or pulmonary condition or that pneumoconiosis materially worsened the impairment caused by cigarette smoking. However, I find that Dr. Castle's medical report refutes Dr. Rasmussen's conclusions and therefore Miner has failed to prove that pneumoconiosis was a substantially contributing cause of his disabling pulmonary impairment.

ORDER

Miner's claim for benefits under the Act is hereby **DENIED**.

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LARRY W. PRICE
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the

[30] Dr. Rasmussen's updated CV may be been included in his 2003 report, which Judge Chapman excluded from the record and was not resubmitted.
[31] I take judicial notice of Dr. Rasmussen's board certification. This information may be found at http://www.abms.org/.

date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).